





*A case of Chronic Tuberculosis of the Nose, Tonsils, Larynx, Trachea, and main Bronchi (sclerous lupus (?) without external manifestations), producing stenosis of the trachea and bronchi. By THOMAS WHIPHAM, M.D., and SHERIDAN DELÉPINE, M.B. Read March 8, 1889.*

GEORGE W., æt. 14, a shop-boy, was admitted into St. George's Hospital under Dr. Whipham on March 15, 1887.

His father died of consumption, but no other member of the family had suffered from this disease, as far as could be ascertained.

The patient had never been strong, but he had never had any serious illness, and neither the previous history nor his present appearance afforded any indication of syphilis. About nine months ago he got wet, and immediately a lump formed on the right side of his throat internally. This "broke" on coughing, but nothing was discharged by the mouth; he had never been well since. He had suffered from cough, but never spat blood. About four or five months ago his voice became weak, and he had been hoarse ever since. He frequently was attacked with paroxysms of dyspnoea of about ten minutes' duration, the attacks occurring chiefly at night. He had slight night sweats, but had not lost flesh. There had been no diarrhoea or vomiting.

On admission the boy was well nourished, his voice weak and husky, and his breathing rather stertorous. There was neither wasting nor night sweats. He had but little cough and not much expectoration. His tongue was clean, his bowels regular as a rule, his pulse 100, not strong, and his respirations 22; his temperature normal. His teeth were very regular and even. Nothing abnormal was found in his lungs, but a loud tracheal stridor accompanied inspiration on both sides. The chest was, if anything, somewhat hyper-resonant.

On examining his mouth the uvula was found to be swollen; both tonsils were enlarged, on the left was a patch

of yellow viscid secretion; the anterior pillars of the fauces were congested. On the right tonsil was a deeply excavated ulcer, and another, more superficial, with a grey base, on the posterior pillar of the fauces on the same side. The larynx generally was congested, but there was no swelling or infiltration of any part of it, save on the right ventricular band, from which an irregular projection passed inwards, and beneath it the vocal cord was very red.

The urine was healthy, of specific gravity of 1018.

Special investigation was made as to any syphilitic taint, either hereditary or acquired, but without eliciting the slightest evidence of the disease. The glands, however, along the posterior margins of both sterno-mastoids and some in the submaxillary region were enlarged.

The treatment was biniodide of mercury, and locally benzoin inhalation, with a liberal diet of meat, milk, eggs, and porter.

Three days later (March 18), in addition to the appearances above described in the mouth and larynx, an oval ulcer, with a yellow base, was discovered, occupying the central third of the right vocal cord, and on the following day the right ventricular band was more swollen, and the ulcer on the cord in consequence partly hidden from view. So far as could be made out, the ulcer had lost its oval shape, and its edges had become irregular in outline.

March 23.—No marked alteration had taken place in the larynx since the last note, except that the right ventricular band was rather more swollen. The ulcer on the posterior pillar of the fauces was larger, but apparently not so deep; it was touched with solid nitrate of silver.

Next day the ulcer on the anterior pillar of the fauces was cleaner and presented a more healthy appearance, but the one behind was larger, irregularly oval, and covered with yellowish white secretion. The gargarisma æruginis of the hospital pharmacopœia was ordered for frequent use. In the larynx the swelling of the right ventricular band was greater, and the ulcer on the vocal cord was in consequence more or less hidden from view.

March 28.—A patch of ulceration was seen on the vomer and inferior turbinated bones, and there was much glandular enlargement in the neck and submaxillary regions. The boy was more anæmic; his breathing was stridulous in character.

On March 31 iron and cod-liver oil were ordered, and the gargle repeated.

April 4.—No improvement had taken place in the larynx; on the contrary, the ulcer on the right cord was larger, and both cords were inflamed, otherwise the patient remained in much the same condition. Morphine and ipecacuanha lozenges were prescribed in addition to the other remedies, and subsequently steam and benzoin inhalations.

During the night of April 17 he was attacked by sudden and alarming dyspnœa, which lasted about half an hour, after which he had no sleep.

On the morning of April 18 the boy complained of shortness of breath, and there was an incessant dry cough. The pharynx and tonsils were still red and congested, but he was too ill for laryngoscopic examination. The tongue was coated with a thin white fur, the papillæ were red and enlarged. He complained too of pain along the anterior border of the trapezius muscle, but nothing abnormal was discovered on inspection and palpation.

April 21.—Abduction of the left vocal cord was performed very sluggishly. The ulceration on the right cord had spread. On the right ventricular band there was an ulcer which had the appearance of a piece having been punched out of the mucous membrane. Both arytenoid cartilages were enlarged, the right the larger of the two.

April 25.—The paroxysms of dyspnœa were more frequent, and he complained of a sensation of phlegm in his throat which he was unable to expectorate. Save that expiration was a little prolonged at the apices of both lungs nothing abnormal was detected.

April 28.—7.30 P.M., tracheotomy was performed in consequence of an urgent attack of dyspnœa, and at 11.30 P.M. the patient became cyanosed in consequence of blocking of the tracheotomy tube. He was ordered 4 oz. of port wine, strong beef-tea, milk, and two eggs.

April 29.—He again complained of obstruction in the tube and had a severe paroxysm of dyspnœa, after which he rallied slightly. This was soon succeeded by a second attack, and in spite of all attempts to clear the tube the patient died at 9.20 A.M.

The autopsy was performed fifteen hours after death. The general condition of the body was good; it was moderately well nourished. On the lower part of the right thigh was a scar  $1\frac{1}{4}$  inches in length. The four upper rings of the trachea had been divided in the operation of tracheotomy.

Permission was only granted to examine the wound and



the parts in the neighbourhood. There were a few scattered soft adhesions in the pleura of the left side. Both lungs were congested but crepitant throughout. At the left extreme apex was a slight puckering and some cicatricial tissue, but nothing like tubercles. At the lower part of the left apex was a calcareous nodule, the size of a small pea, surrounded by pigment and contained in the pleura. The bronchial glands on the left side were hard, and one was calcareous. At the base were a few calcareous plates, thin and flat, in the pleura. The right lung showed no puckering, cicatrices, or any trace of tubercle. In the pleura there were numerous flat calcareous plates ranging in size from a pin's head to a pea, and each surrounded by a zone of black pigment; these plates were much more numerous on the right than on the left side.

*Larynx.*—There was no thickening of the epiglottis or arytenoids. On the right vocal cord was a small papillomatous growth. Between the right vocal cord and the epiglottis was an ulcer involving the vocal cord, ventricular band, and ventricle of the larynx.

*Trachea.*—Below the fourth ring of the trachea its calibre was much constricted by submucous thickening, chiefly on the right side posteriorly. The mucous membrane was red below the wound and showed one small ulcer. The submucous stenosis, which was excessive in the lower part of the trachea, extended down the right bronchus and into the smaller bronchial tubes of the right side. The left bronchus and its larger subdivisions were much dilated, the primary left bronchus being of twice its normal calibre. The vessels show no thickening. To the back of the first bifurcation of the right bronchus a small black bronchial gland was adherent.

Three specimens were removed for microscopical examination:

1. The first included a portion of the ulcer found above and over the anterior portion of the right vocal cord, in the region of the fossa centralis.

2. The second was a complete thin transverse slice of the trachea at the level of the ninth cartilaginous ring. At that point the lumen of the trachea was extremely reduced, it measured about 7 mm. in its greatest width, and its longest antero-posterior diameter was not more than 5 mm. (after hardening in spirit).

3. The third specimen was taken from the right bronchus,

and consisted of a transverse slice through the inner and posterior half of the bronchus, about half an inch below the bifurcation of the trachea. This section included a small calcified nodule adhering to the bronchus at that level.

### 1. *Laryngeal Ulcer.*

(a) *General description.*—The true vocal cord, the subglottic mucosa and submucosa, the mucosa of the anterior portion of the ventricular opening, the thyro-arytenoideus muscle, are exposed in the sections which are perpendicular to the vocal cord.

Above the true vocal cord the mucosa is much swollen, infiltrated with small cells, and in some places is beginning to ulcerate. Numerous tubercular nodules are found in the deeper parts of this infiltrated membrane.

The vocal cord itself is invaded by the granulation tissue.

The subglottic mucosa is thickened, its mucous glands are partly destroyed, there is much small-cell infiltration around what remains of some of these glands.

The epithelial lining of the larynx below the glottis is much altered and thickened; above it is altered also and partly necrosed, over the vocal cord it is in a state of catarrhal desquamation.

The thyro-arytenoideus muscle is in a state of progressive interstitial myositis leading to gradual atrophy.

Some of the deeper vessels are obstructed either through endarteritis or through thrombosis.

Many of the lymphatic vessels or spaces are distended with coagulated hyaline or granular lymph. Some of the same material is found also effused just under the epithelium on the surface of the granulation tissue replacing the mucosa.

(b) *Detailed description.*—1. *The epithelium* is distinctly stratified squamous above and below the vocal cord. Over the vocal cord the superficial layers of the epithelium have desquamated, so that the deep layers only remain attached to the basement membrane in the form of pyramidal and pyriform cells more or less isolated one from the other. Thus the vocal cord seems to be covered with a modified columnar epithelium (the result of inflammatory changes), whilst above and below the cord the mucous membrane is covered with a stratified squamous epithelium.

In several places above the vocal cord the epithelium has entirely lost its structure and is evidently necrosed ; at two or three points it is entirely destroyed, leaving the subjacent infiltrated mucosa exposed.

In some regions the epithelium is separated from the underlying structures by an accumulation of translucent coagulated material, parts of which look very granular and as if composed of micro-organisms when examined with an ordinary power. After staining by Gram's and other methods only a few putrefactive bacteria can be discovered in these masses ; a great part of the granular appearance is apparently due to the precipitation of fibrin. A few small hæmorrhages can also be found just under the epithelium.

2 and 3. *Mucosa and submucosa.*—Owing to the changes they have undergone these two layers cannot be distinguished, and must be taken together. Above the vocal cord they are almost entirely replaced by granulation tissue ; the superficial parts are chiefly composed of young vessels, mostly running perpendicularly to the surface, and separated by small cells.

The deeper parts are occupied by an almost continuous layer of tubercular nodules with very distinct giant-cells.

The *subglottic mucosa* is much less infiltrated than the ventricular mucosa ; it contains, however, a large number of dilated small vessels, and is composed of an unusually transparent form of areolar fibrous tissue. This fibrous tissue has evidently undergone some form of hyaline degeneration. Through this tissue the narrowed ducts of the more or less atrophied mucous glands make their way to the surface. The almost entire absence of mucous glands indicate that a considerable number of these glands must have been destroyed. Those that remain are practically useless. Some are embedded in the modified fibrous tissue above described, and in that case their acini are often reduced to mere specks. Others are surrounded by small cells which permeate the whole of their stroma, and in that case the epithelium of the acini is in one or other of the states indicating parenchymatous inflammation.

The *inferior thyro-arytenoid ligament* is much infiltrated, in fact the edge of the tubercular infiltration coincides with its upper border. Many of its elastic fibres are altered.

The *thyro-arytenoideus* muscle is much affected, but mostly at the level of the vocal cord, where a large number of its fibres is found to be in an advanced state of atrophy, and the



endomysium and perimysium much increased in amount. This is of course seen chiefly on the internal aspect of the muscle.

No bacillus tuberculosis could be found in these sections, although a very large number (fifty at least) of sections were stained for that purpose. It is well to state, however, that the specimen had been hardened in chromic acid.

## 2. *Trachea.*

(a) *General description.*—The lumen is much diminished and has an irregular trapezoidal outline. This is due to the fact that the thickening of the mucosa and submucosa is most marked anteriorly, posteriorly and at the sides, and that there is on the contrary contraction on the intervening regions.

The *epithelium* is stratified, squamous, and irregularly thickened; it is destroyed in a few places.

The *mucosa and submucosa* are much altered, and can hardly be recognised one from the other, being both almost entirely composed of a very hyaline form of connective tissue already described, with patches of lymphoid-looking tissue with or without giant-cells. The superficial layers of the mucosa are replaced by a stratum of granulation tissue, varying much in thickness. Small hæmorrhages, and masses of coagulated lymph, are found here as in the larynx.

The *mucous glands* are entirely destroyed.

The *cartilages* are in a state of chronic inflammation leading to their slow destruction, replacement by fibrous tissue, and dislocation.

The *adventitious layers* of the trachea are in the same state as the submucosa.

The *adipose tissue* is slowly replaced by lymphoid tissue with or without giant-cells, or by the hyaline areolar fibrous tissue described above. The *vessels* are in a state of endarteritis obliterans, and the *nerves* show the changes generally observed in interstitial neuritis.

(b) *Detailed description.*—The epithelium, as we have already noticed, is remarkably altered, and has taken all the characters of a stratified squamous epithelium such as is normally found in the mouth. The cells show most distinctly the prickly appearance usually observed in the rete Malpighii of that form of epithelium. [This remarkable instance of metaplasia is the best proof that has fallen under my notice of the homo-

logy between the deep layers of the rete Malpighii and ciliated epithelia—a fact which I have tried to establish a few years ago and to which I intend to refer in some further paper based on this case—S. D.] This epithelium is very thick at the places where the mucous membrane is thinnest, namely at the four angles of the trapezoid lumen above described. There it sends processes into the subjacent mucosa, which forms a number of papilliform projections between these epithelial digitations. Where the mucous membrane is thicker the epithelium is on the contrary thinner. In some regions it is very thin, at places it is raised from the surface of the mucosa by subepithelial effusion of blood or of coagulated lymph. It is even absent over a few small areas.

In the *mucosa* and *submucosa* we have already noticed a very great alteration of structure, and an entire absence of mucous glands. We have also directed attention to the considerable difference of thickness that exists between the various parts; this thickness varies between one twelfth and one fourth of an inch (after hardening). [It is probable that during life greater differences must have existed, owing to the temporary swellings that must have resulted from the congestion, effusions, or hæmorrhages which can be noticed in various parts of the specimen.]

The structure of these membranes is not entirely the same in the thick and in the thin parts.

In the thick parts there is generally a greater amount of lymphoid or granulation tissue under the epithelium, and also a greater number of patches of cellular infiltration in the deeper parts, than is found in those parts where the membrane is thin. In the regions where the mucosa and submucosa form a thin layer, there is on the contrary a greater amount of rather dense and cicatricial-looking tissue. In the region where the mucosa and submucosa form a thick layer, the parts intervening between the cellular patches have an areolar arrangement; but the intercellular substance, instead of having the appearance of wavy bundles of fine fibrils, has a very transparent and homogeneous look and is evidently in a state of hyaline degeneration. In some places the homogeneous intercellular substance forms a close network with one or two cells only in each mesh, and there the modified tissue looks more as if it had resulted from the transformation of lymphoid tissue, the alteration resulting from hyaline degeneration and swelling of the reticulum.

The changes observable in the *cartilaginous rings*, the

*adventitious layers*, the *vessels* and the *nerves* have already been alluded to.

### *Right Bronchus.*

This bronchus is much less stenosed than the trachea. The *epithelium* is less altered, but instead of being columnar ciliated as it should be, it belongs in many places to a low cubical type.

The *mucosa* is much congested, but comparatively little infiltrated with small round cells.

The *submucosa* can here be recognised, but is much altered, chiefly in the neighbourhood of the *mucous glands*, which are in a more or less advanced state of parenchymatous and interstitial inflammation and atrophy, complete destruction having occurred in some of them. The *cartilages* are also much altered, chiefly on their inner aspect; and here the multiplication of the cells, their alteration in size and shape, as well as the fibrous transformation of the matrix, leading to the destruction of the cartilage as such (*metaplasia*), are all very well seen.

The *adventitious layers* are here less altered than in the case of the trachea.

The *calcified bronchial gland* is a very small one, measuring about  $\frac{1}{2}$  inch by  $\frac{1}{4}$  inch. It is surrounded by a thick capsule of fibrous tissue arranged in concentric layers.

A number of trabeculæ divide the nodule into a number of small spaces each containing an irregular calcareous mass. In the trabeculæ the lymphoid structure may still be recognised, but it is much altered by chronic inflammatory changes. Many giant cells, as well as a number of pretty large cells of various shapes, are found in this altered tissue, but no distinct tubercular nodule can be discovered either in or about the gland.

### *Conclusions.*

The lesions found in this case are, some of them, so characteristic that it is hardly possible to doubt their tubercular nature, and therefore *primâ facie* the case seems to be one of chronic tubercular affection of the *nose*, *tonsils*, *larynx*, *trachea*, and *bronchi*. It further seems probable that the case is more closely allied to *lupus* than to any other variety of lesion produced by tuberculosis.



Before coming to a definite conclusion it seems necessary, however, to set aside a certain number of serious objections.

*First objection.*—There is hardly any well-proved case of tubercular stenosis of the trachea on record.

Rokitansky in one case was unable to say whether the cause of constriction was tuberculosis or syphilis or a mixture of the two diseases.

Lemcke mentions another case in which a patient, having suffered repeatedly from hæmoptysis, became affected with cylindrical stenosis of the larynx, but in this case the man recovered at the age of forty-eight.

Sopel gives a case in which stenosis of the trachea and bronchi was due (he supposes) to the extension of the tubercular process from peribronchial and peritracheal glands to the walls of the air-passages, but in this case carcinoma of the thyroid body was also found post mortem.

Dr. Bateman has just recorded another instance of laryngeal stenosis which he thinks may be tubercular, but in this case there was no distinct history of tuberculosis, and no direct evidence that the process was tubercular.

*Second objection.*—Dr. Dubar says, basing his assertion on the study of a large number of cases of stenosis of the trachea published up to 1884, that he has not been able to find a single case where tuberculosis has given rise to stenosis (that is, that all cases recorded were supposed to be due to some other cause).

*Third objection.*—The seat of the lesion is more like what is found generally in syphilis than in tubercular disease. Thus Dubar found in eighteen cases of syphilitic stenosis of the trachea which he had been able to collect, that

In 77 per cent. the lower fourth was affected.

In 11 „ the whole length was affected.

In 6 „ the middle part was affected.

In 6 „ the upper part was affected.

Whilst in 33 per cent. of all these cases the bronchi were also affected.

*Fourth objection.*—The changes observed in the epithelium have not been noticed before except at the margin of syphilitic ulcers (Ziegler).

*Fifth objection.*—The excessive formation of cicatricial tissue, and the destruction of pre-existing tissues, are more commonly found in syphilis of the trachea than in any other affection of that organ. The entire destruction of the glands



is more like what is found in cases of leprosy, and in many parts the structure of the mucous membrane agrees remarkably with some of the descriptions given of rhinoscleroma.

*Sixth objection.*—We have been unable to discover any *Bacillus tuberculosis* in a large number of sections taken from various parts of the organ. [I examined at least fifty preparations, in the staining of which I used various preparations of ordinary fuchsin and of rubin coming from the best sources. At my request Dr. Slater was kind enough to examine also a large number of sections, and was equally unsuccessful.—S. D.]

In presence of all these difficulties it is necessary to discuss the possibility of any other explanation of the case.

*Stenosis of the larynx or trachea* (we suppose that most processes affecting the mucous membrane of the larynx may also affect that of the trachea by extension) may be due to :

1. *Chronic non-specific inflammation, traumatism* (Gintrac, Andral, Trousseau, Demarquay, Gibb, Wilks, &c.).

2. *Syphilis* (Worthington, Moissenet, Charnal, Boeckel, Payne, Lancereaux, Norton, Thornton, Cornil, Beger, Oudin, Berger, Morell Mackenzie, Jacobson, Dubar, Frankel).

3. *Tuberculosis* (Rokitansky?, Sipel?, Lemcke?, Bateman?).

4. *A mixture of syphilis and tuberculosis* (Rokitansky, Schnitzler, Arnold).

5. *Lupus* (Türck, Tobold, Ziemssen, Grossmann, Lefferts, Morell Mackenzie, Cornil and Ranvier, Babes, Kaposi).

6. *Leprosy* (Morell Mackenzie, Cornil and Ranvier, Babes, Wolff, Gibb, Schroetter, Elsberg).

7. *Rhinoscleroma* (Chiari, Hebra, Billroth, Kaposi, Pellizzari, Cornil and Alvarez).

8. *Chronic glanders* (Tardieu, Dubar).

(A few authorities only are given in support of these statements, but a great many more could easily be found.)

The presence of distinct granulomata in the diseased tissues allows us at once to put aside *simple inflammatory thickening*. It is equally evident that we have no indication in the case of the existence of *leprosy* or *glanders*.

There remains, therefore, only *syphilis*, *tuberculosis*, and *rhinoscleroma* to discuss.

We have already pointed out most of the features which give to the lesions a syphilitic appearance. Against this view we have the following facts :

1. There is no history of acquired syphilis, which, according to Rindfleisch, is the disease which generally gives rise to

ulceration and stenosis. There is no history and no indication of hereditary syphilis.

2. There are none of the deep and typical ulcers generally found in these cases at the point of constriction (Charnal, Verneuil, Norton, Payne, Lancereaux, Beger, Dudin, Dubar).

3. The glands are entirely atrophied, whilst, according to Dubar, they should in syphilis be rather enlarged and distended with mucus.

4. Although the presence of a few patches of stratified squamous epithelium at the border of syphilitic ulcers is mentioned in Ziegler's text-book of pathological anatomy, we are not aware that any such extensive alteration of the structure of the epithelial lining as is found in this case has been mentioned in connection with any syphilitic or other affection of the trachea before this.

5. The presence of tubercular nodules in the larynx and trachea could only be accounted for by supposing that tuberculosis and syphilis were concomitant in this case, but the presence of tubercles at various levels, and in the midst of the cicatricial tissue in all the parts of the trachea, but specially in those regions where the tissue changes seem to be progressing, would imply a perfectly parallel extension of the two processes, an hypothesis difficult to accept without absolute proof.

6. In syphilitic cases stenosis is the result of cicatricial contraction following ulceration, while in this case it seems evident that ulceration, where it exists at all, is very superficial and secondary to the processes taking place in the deeper strata.

These objections to the syphilitic hypothesis are certainly of greater importance than those made against the tubercular one.

It has been incidentally mentioned that the structure of the newly-formed tissues of some parts of the trachea reminds one of the structure described by Cornil and Alvarez in connection with rhinoscleroma of other parts.

Chiari has written a paper on stenosis of the trachea due to rhinoscleroma, and the possibility of the extension of the disease to the larynx is mentioned in several works.

There are, however, many points of difference between this case and those of rhinoscleroma which have been recorded.

1. The history of the case, which has nothing in common with that of rhinoscleroma.

2. The situation of the point of greatest constriction.
3. The great changes observed in the epithelium.
4. The destruction of the glands.
5. The presence of very distinct tubercular nodules on the larynx. (It must be said, however, that the arrangement of the lymphoid tissue and of the giant-cells in the trachea is in most places very irregular, and that a well-formed tubercle is rare in the lower part of the trachea.)

6. The very small number of the hyaline globules described so carefully by Cornil and Alvarez.

Without discussing the possibility of a diffuse lymphomatous growth, for which there is no foundation, there remains now to answer the objections which may be raised against tuberculosis, including lupus.

1. Although there is no authentic case of tubercular stenosis of the trachea, there are two very interesting cases of lupus of the larynx, published by Dr. Michael Grossmann, of Vienna, which both bear remarkably upon this case.

In one the patient, a boy, living under unfavorable circumstances, had a swelling in the left submaxillary region when he was seven years of age; suppuration and ulceration took place and the border of the ulcer assumed the appearance of lupus vulgaris, the process extended externally, involving the whole skin of the region. Owing to dyspnoea caused by swelling in the larynx, cauterization with lactic acid was resorted to with good effect. The boy was ten years old in 1887, when the case was published.

In the other case, that of a female patient who had always been hoarse, there was lupus of the conjunctiva, nose, and upper lip, and in addition there was a large heart-shaped defect in the central portion of the epiglottis. The vocal and ventricular bands were covered with a moderate amount of granulation tissue, of which a small mass was also found beneath the anterior commissure of the vocal cords. There was also partial distortion of the uvula. After ten years' observation only slight implication of the hard and soft palate and pharynx had taken place.

Thus in the first case we have a swelling in the submaxillary region, whilst in the case under discussion there was, nine months before admission, a swelling on the right side of the throat. In both cases the swelling burst; only in one case the swelling opened externally, whilst in the other it broke internally. In both cases dyspnoea and other throat



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symptoms supervened after a time. Both patients were young, and were of weak constitution.

In the second of Dr. Grossmann's cases we have a distinct account of granulations covering the vocal cords and rendering them uneven, and of the same kind of lesion in the region of the anterior commissure of the vocal bands.

2. The objection based on Dubar's statement has, of course, no more value than objections based on similar grounds. It is not because a lesion has not been identified that it has never existed.

3. The same answer can be given with regard to the unusual seat of the lesion. The case of Rokitansky is interesting in that respect, for it shows that, in some cases, at least pathologists may have been in doubt regarding the nature of certain lesions which may have been recorded as syphilitic because there was no distinct evidence that they were of other origin.

4. As to the changes observed in the epithelium, they seem to result from two causes, (a) unusual irritation, (b) dryness of the membrane, due to destruction of the mucous glands. A similar stratification of the epithelium is observed in certain acute inflammations of the air-passages, but in those cases, the proliferation of the epithelium taking place rapidly, the cells have no time to undergo the changes leading to the formation of definite squamous and spinous cells. On the other hand, it is well known that mucous membranes exposed to the direct drying influence of the external air often become covered with stratified squamous epithelium. Given any process that will lead to irritation of the epithelium and drying of its surface, or possibly the latter only, we may expect as a result the formation of a stratified squamous epithelium. We may therefore safely assume that the state of the epithelium has nothing specific in this case, and is the simple result of the changes which have taken place in the subjacent strata. Hence it is reasonable to discard entirely this objection. On the other hand, it is well known that great hypertrophy of the rete Malpighii is not an unknown feature in tubercular lupus of the skin.

5. The excessive formation of cicatricial tissue in the trachea can hardly be said to be a serious objection; the formation of fibrous tissue is one of the commonest results of chronic tuberculosis, and that this formation has not been before observed in the trachea to the same extent as in the present case is no insuperable objection. Then we are accustomed to consider at least some forms of lupus as distinctly



tubercular, and in certain cases of lupus the abundant formation of dense fibrous tissue is a very important feature of the process. (See Kaposi and Vidal and Leloir.)

6. Our unsuccessful attempts at staining the *Bacillus tuberculosis* in this case is certainly a strong objection, but it must be remembered that all observers agree on the great difficulty of demonstrating the presence of the bacillus in many cases of lupus. Koch himself has obtained positive results only by staining a very large number of sections in each case which he has studied. Owing to an unfortunate circumstance, the chances of discovering the organism have also been greatly diminished by the specimen having been left for seven days in weak chromic acid, and this, as is well known, renders the staining process unusually difficult.

7. We have also to bear in mind the distinct tubercular history of the patient.

After weighing all these matters carefully, and keeping in mind the absence of pyrexia, it seems evident that, notwithstanding the absence of any very distinct antecedent, the case must be considered as one of chronic tuberculosis of the larynx, trachea, and bronchi, probably a form analogous to sclerous lupus, without any external manifestation.

The diagnosis of lupus is also supported by the absence of distinct active tubercular affection of the lungs, and the very large number of giant-cells in the tubercles of the trachea. (Some of the superficial lesions due to hæmorrhage, exudation, and small-cell infiltration, which seem to result from a recent irritative process, must have been the result of the exacerbation which took place some time before, and ended in the death of the patient.)

*Publications referred to, directly or indirectly, in the discussion of the pathology of this case.*

No.	Date.	Author.	Reference.	Nature of publication.
1	1834	Andral.....	<i>Clinique médicale</i> , iii, p. 183	Inflammatory stenosis.
2	1835	Reynaud .....	<i>Mém. Acad. de Méd.</i> , iv, p. 117	Obliteration of bronchi.
3	1843	Worthington .....	<i>Med.-Chir. Trans.</i> , xxv	1 case of syphilitic stenosis.
4	1843	A. Tardieu .....	<i>Thèse</i> , Paris	Chronic glanders, causing stenosis.
5	1844	E. Gintrac .....	<i>Bullet. méd. Bordeaux</i> (June)	Inflammatory stenosis.

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No.	Date.	Author.	Reference.	Nature of publication.
6	1858	Moissenet .....	<i>Union médicale</i> , Oct. 28	1 case of syphilitic stenosis.
7	1859	Türck .....	<i>Zeitsch. d. Gesellsch. d. Aerzte zu Wien.</i> , 1859, No. 11	5 cases of lupus of the larynx.
8	1859	Charnal .....	<i>Union médicale</i> , Feb. 19	1 case of syphilitic stenosis.
9	1859	Charnal .....	<i>Thèse</i> , Paris	Remarks on stenosis of trachea following syphilitic ulceration.
10	1859	Huguier .....	<i>Bullet. de la Soc. de Chirurgie</i> , p. 534	Case of syphilitic stenosis cured by treatment.
11	1862	Empis .....	<i>Du Cornage</i>	Roaring, whistling, wheezing as symptoms of stenosis of trachea.
12	1863	Wagner .....	"Das Syphilom," <i>Arch. der Heilkunde</i> , p. 121	Syphilis as a cause of stenosis of trachea.
13	1863	Boeckel .....	<i>Soc. de Chir.</i> , Dec.	1 case of syphilitic stenosis.
14	1864	Baudré .....	<i>Thèse</i> , Paris	Stenosis of the trachea.
15	1864	E. Vidal .....	<i>Bullet. et Mém. Soc. Méd. des Hôp.</i> , p. 139	Cure of a case of syphilitic stenosis of trachea.
16	1865	Mary .....	<i>Thèse</i> , No. 148, Paris	On stenosis of air passages.
17	1866	J. Cyr .....	<i>Thèse</i> , Paris	Pathological anatomy of stenosis of trachea.
18	1866	Türck .....	<i>Klinik. d. Krankh. d. Kehlkopfer</i>	Syphilitic disease of trachea.
19	1867	Gerhardt.....	<i>Deut. Arch. f. klin. Med.</i> , ii, iii	Syphilitic disease of trachea.
20	1868	Raynaud (M.).....	<i>Bull. Soc. Anat.</i> , p. 349	Cicatricial stenosis of trachea.
21	1869	A. Després .....	<i>Soc. Chirurg.</i> , p. 175	Syphilitic stenosis; cure.
22	1869	Trélat .....	<i>Bullet. de l'Acad. de Méd.</i> , xxxiv, p. 190	Indications and results of tracheotomy in syphilitic lesions of air passages.
23	1869	Payne .....	L. Dubar (see)	Histology of syphilitic ulcer.
24	1870	Morell Mackenzie	<i>Pathol. Trans.</i>	1 case of syphilitic stenosis.
25	1871	Lancereaux.....	<i>Atlas d'Anat. Path.</i>	
26	—	—	<i>Traité Hist. et Prat. de la Syph.</i>	1 case of syphilitic stenosis.
27	1872	Norton .....	<i>Pathol. Trans.</i>	1 case of syphilitic stenosis.
28	1874	Thornton .....	<i>Pathol. Trans. and Lancet</i> , June	1 case of syphilitic stenosis
29	1875	Hebra and Kaposi	<i>On Diseases of the Skin</i> , pp. 89 to 98, chiefly 92 and 95	Anatomy of lupus. Hypertrophic lupus, &c.
30	1875	Cognes.....	<i>Thèse</i> , Paris	Roaring in mau.
31	1875	J. L. Binet.....	<i>Thèse</i> , Paris	Broncho-tracheal roaring and its relation to sudden death.
32	1871	Schrötter .....	<i>Jahresbericht der Klin. für Laryngoscopie</i>	Stenosis of trachea.
33	1875	—	<i>Laryngolog. Mittheilungen</i> , p. 102	Stenosis of trachea.

No.	Date.	Author.	Reference.	Nature of publication.
34	1876	Daret .....	<i>Arch. gén. de Méd.</i> , 6e serie, i, pp. 578—715	Stenosis and distortion of trachea.
35	1876	F. Riegel.....	<i>Berlin. klin. Wochenschr.</i> , No. 47, p. 673	Symptomatology of stenosis of air passages.
36	1877	Grossmann .....	<i>Wien. med. Zeitung</i> , No. xx	Lupus of the larynx, 1 case.
37	1878	A. Vierling .....	<i>Deutsches Arch. für klin. Med.</i> , p. 326	Syphilis of trachea and bronchi.
38	1878	Lefferts .....	<i>Amer. Journ. of Med. Science</i> , April	1 case of lupus of larynx.
39	1879	Cornil .....	<i>Leçons sur la Syphilis</i>	Histology of cicatricial thickening.
40	1879	Beger .....	<i>Deutsches Arch. f. klin. Med.</i> , xxiii, pp. 608—614	2 cases of syphilitic stenosis.
41	1880	Morell Mackenzie	<i>Disease of Throat and Nose</i> , vol. i, p. 396	2 cases of lupus of larynx, nose, lips.
42	1880	Braun .....	<i>Centralblatt für Chir.</i> , No. 51	Cure of stenosis of larynx and trachea by dilatation by means of india-rubber bag.
43	1880	Oudin .....	<i>Soc. Anatomique</i> , Nov. 5	} 1 case of syphilitic stenosis.
	1881	— .....	<i>Progrès Médical</i> , p. 343	
44	1881	W. Berger .....	<i>Schmidt's Jahrb.</i> , excii, 144	Syphilitic stenosis of air passages.
45	1881	J. N. Mackenzie...	<i>Wien. med. Jahrb.</i> , p. 75	1 case of syphilitic stenosis.
46	1881	Morell Mackenzie	<i>Trans. Path. Soc.</i> , xxii	Syphilitic disease of trachea.
47	1881	Jacobson .....	<i>Samml. klin. Vorträge</i> , herausg. von R. Volkmann, No. 205; <i>Innere. Med.</i> , No. 68	Syphilitic stenosis, 1 case.
48	1882	Chiari .....	<i>Medicin. Jahrbucher von der k. k. Gesellschaft der Aertze</i> , Heft 2	Stenosis of larynx and trachea due to rhinoscleroma.
49	1882	Cornil and Ranvier	<i>Manuel d'Histolog. Pathologique</i> , ii, pp. 51, 845	Syphilis of trachea.
50	1882	Lancereaux.....	<i>Annales des Maladies de l'Oreille et du Larynx</i> , p. 117	Lupus.
51	1883	Cornil .....	<i>Soc. Anatomique</i> , p. 319	1 case of syphilitic stenosis.
52	1884	L. Dubar.....	<i>Nouveau Dict. de Méd. et de Chir. Prat.</i> (Jacquoud), xxxvi	Rhinoscleroma.
53	1885	Cornil and Alvarez	<i>Archiv. de Physiologie</i> , 3e ser., vi, p. 11	Stenosis of the trachea.
54	1885	Vidal and Leloir...	<i>Traité de Pathologie cutanée</i>	Rhinoscleroma.
55	1886	Cornil and Babes...	<i>Les Bacteries</i> , pp. 650, 738	Lupus.
56	1886	E. Ziegler .....	<i>Special Path. Anat.</i> , iii, 106	Syphilitic disease of trachea.



No.	Date.	Authors.	Reference.	Nature of publication.
57	1887	E. Frankel .....	<i>Deut. med. Woch.</i>	Peculiar density of cicatricial tissue in stenosis of trachea, 1 case.
58	1887	Sopel .....	<i>Annales des Maladies de l'Oreille</i>	Tubercular steuosis of trachea, 1 case.
59	1887	M. Grossmann ...	<i>Med. Jahrb. der k. k. Gesell.</i>	Lupus of the larynx, 2 cases.
60	1887	Schnitzler .....	<i>Inter. klin. Rundschau.</i>	Syphilitic ulcers a good nidus for <i>Bacillus tuberculosis</i> .
61	1888	Arnold.....	<i>Pacif. Med. and Surg. Journ.</i>	Case of concomitant tuberculosis and syphilis of larynx.
62	1888	Chr. Lemcke .....	<i>Berlin. klin. Woch.</i> , 13, p. 247	1 case of tubercular stenosis (?)
63	1888	Ed. Rindfleisch ...	<i>Path. Hist.</i> (French translation), p. 517	Acquired pulmonary syphilis.
64	1889	F. Bateman.....	<i>Brit. Med. Journ.</i> , Jan 5	Obscure case of laryngeal steuosis.
65	1888	Solis Cohen.....	<i>Annual of Universal Med. Sciences</i> , iii, 822, Sajous.	Several cases of stenosis, syphilitic and tubercular.
66	1888	J. Bex ... ..	<i>Rev. de Scien. Méd.</i> , xxxii, 284	Case of tubercular stenosis (see Lemcke).

The case is submitted to the Society as being of much clinical and pathological interest, and also because we have been unable to find, by reference to the literature of the subject, a similar case.

In its clinical aspect the case was obscure from first to last. The appearance of the ulcers when the boy first came under observation was very much that of syphilis, and even after a most careful inquiry as to the probability or possibility of the disease having been acquired had failed to elicit any evidence of such infection, it was difficult to relinquish the idea that after all the disease was syphilitic. Mercury, however, produced no beneficial effect, and this diagnosis was consequently abandoned. The patient was then treated with steel wine and cod-liver oil under the assumption that it was, in spite of the absence of any signs or symptoms of pulmonary affection, of a tubercular nature.

Even so there were important clinical facts which ranged themselves rather against than in favour of such a diagnosis; that is to say that from March 15 until April 29, while the boy was under treatment, the temperature never exceeded



100° F., and reached that point on one occasion only ; there was no cough, no expectoration, no night sweating, no albuminuria, and but little, if any, loss of flesh, and lastly there were no signs of pulmonary disease.

Until lately it was held that laryngeal or tracheal affections of a tubercular nature were always accompanied by disease of the lung, and as recently as 1879 Heinze, in his well-known essay, states definitely that ulceration is never found with tuberculosis of other organs without the lungs being affected, and he bases his statement upon 4486 consecutive autopsies.

Demme, however, in 1883 recorded the case of a child, æt. 4½, who died of tubercular meningitis, and in whom laryngeal ulceration was found with the bacilli of tubercle, the thoracic and abdominal organs being free from tubercular disease. In the interval one or two similar cases have been placed on record, thus establishing the fact that, although of rare occurrence, the condition may and does occasionally exist.

*Report of a Committee appointed by the Society to examine the foregoing case.*

We have examined the specimen of tubercular lupus of the trachea exhibited by Drs. Whipham and Delépine, and are of opinion that there is undoubted evidence of the tubercular nature of the disease. Further, we are of opinion that there is not sufficient evidence to prove that the conditions exhibited in the specimen were due to any other disease in addition to tuberculosis. We therefore confirm the view which has been expressed and excellently supported by the exhibitors.

HENRY T. BUTLIN.

W. WATSON CHEYNE.

PERCY KIDD.

THOMAS WHIPHAM.

SHERIDAN DELÉPINE.





## DESCRIPTION OF PLATE VI.

Drs. Whipham's and Delépine's case of Tubercle of the Larynx and Trachea.

FIG. 1.—General appearance of the larynx, trachea opened from behind.

- (a) Laryngeal ulcer.
- (b) Altered central fossa.
- (c) Laryngotomy wound.
- (d) Thickened mucous and submucous membranes.
- (e, e') Transverse section of the trachea, showing the thickened coats and angular lumen.
- (f) Dilated left bronchus (extra-pulmonary bronchiectasis).
- (g) Calcified gland attached to right bronchus.

FIG. 2.—Transverse section of the trachea at the level of the ninth cartilaginous ring.

- (a) Sclerosed and hyaline adventitious coat.
- (b) Small granuloma (tubercular).
- (c) Small portion of cartilaginous ring separated from the rest.
- (d) Fibrous tissue which has replaced part of the cartilaginous ring.
- (e) Sclerosed and hyaline mucosa and submucosa (notice the great thickness of these membranes and the absence of mucous glands).
- (f) Thin and contracted portion of these membranes owing to that contraction; the adjacent portion of the cartilaginous ring has become dislocated.
- (g) Epithelium where it is thin and atrophied.
- (h) Epithelium where it is thick and sends interpapillary processes into the subjacent mucosa.
- (i) Sclerosed and atrophied fatty lobules in the adventitia.



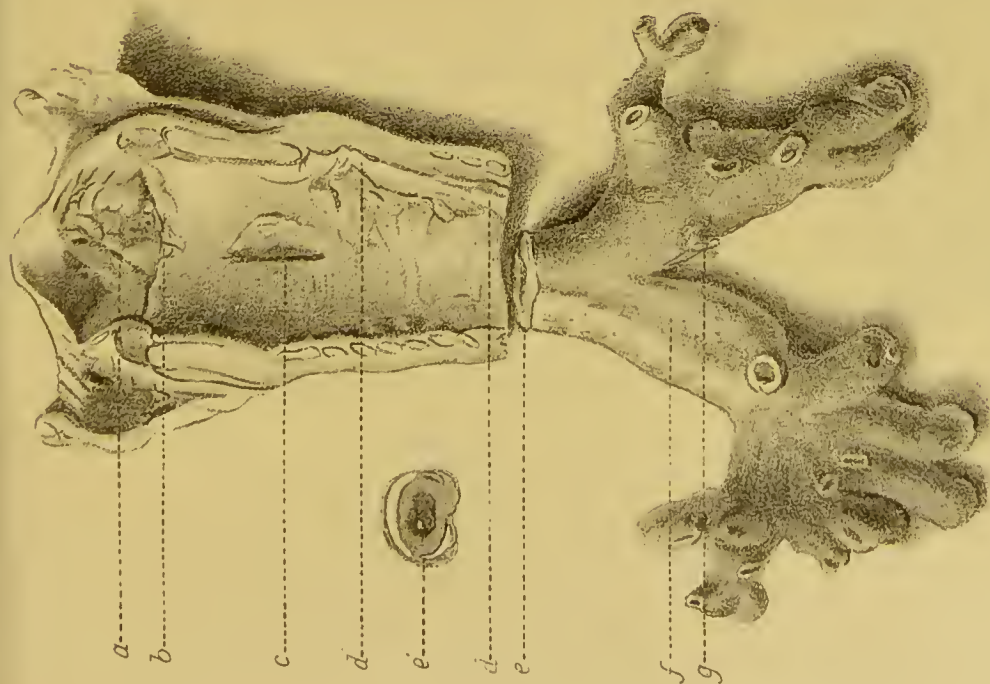


FIG. 1

Sheridan Delepine, del.

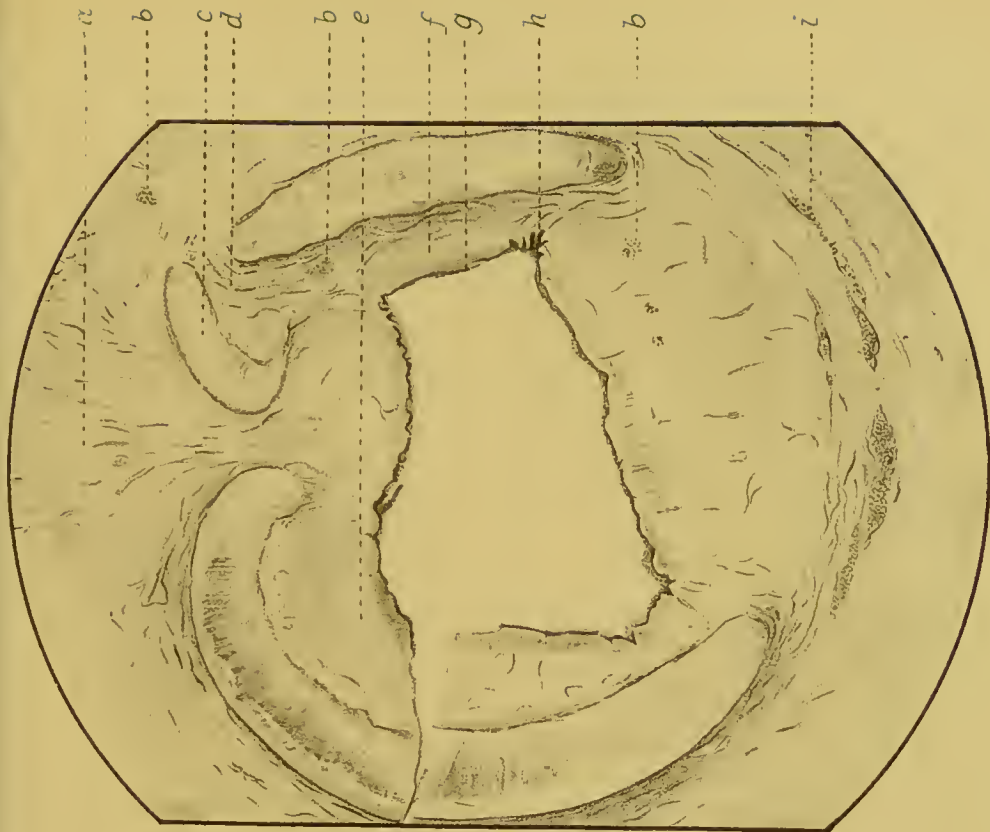


FIG. 2.

Danielsson & Co. lith.





## DESCRIPTION OF PLATE VII.

Drs. Whipham's and Delépine's case of Tubercular Disease of the Larynx and Trachea.

FIG. 1.—Right vocal cord (anterior part, near anterior commissure).  
× 24.

- (a) Necrosed epithelium, covering
- (b) Granulation tissue.
- (c) Ducts of mucus glands.
- (d) Tubercular nodules.
- (e) Giant-cells.
- (f) } Muscular bundles, atrophied from interstitial myo-
- (g) } sitis.

FIG. 2.—From right bronchus. × 24.

- (a) Epithelial lining and infiltrated mucosa.
- (b) Mucus glands, infiltrated and inflamed.
- (c) Superficial layers of cartilaginous rings with commencing erosion.
- (d) Deeper layers.
- (e) Adipose tissue.
- (f) Sclerosed connective tissue.
- (g) Thickened capsule of
- (h) Lymphatic gland, containing calcareous masses.





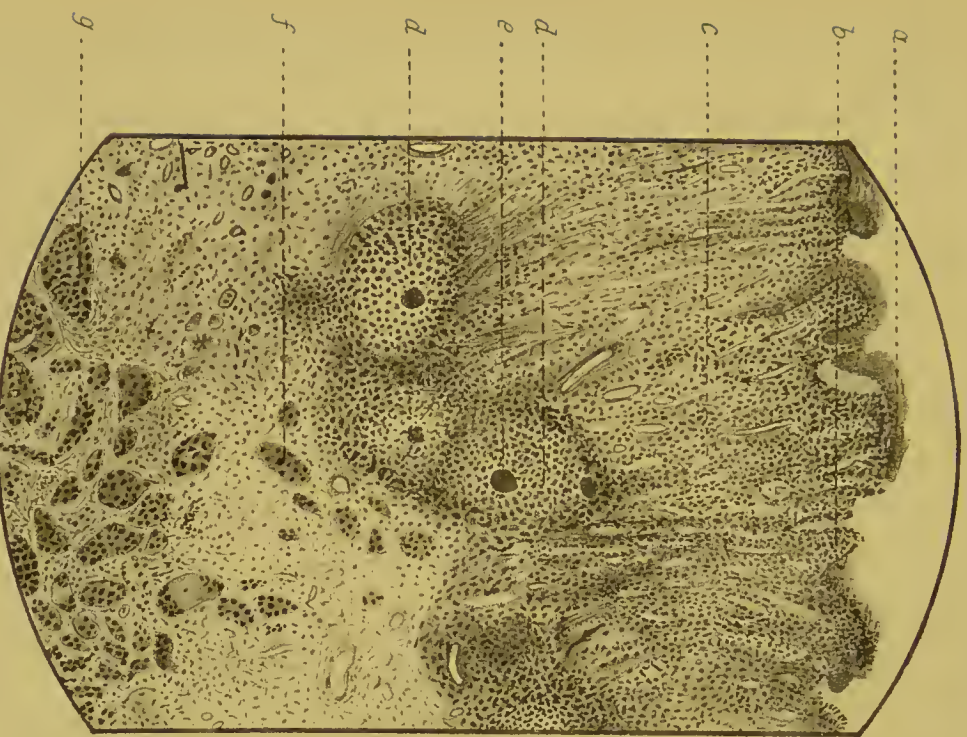


Fig. 1.

Sheridan Delepine dr

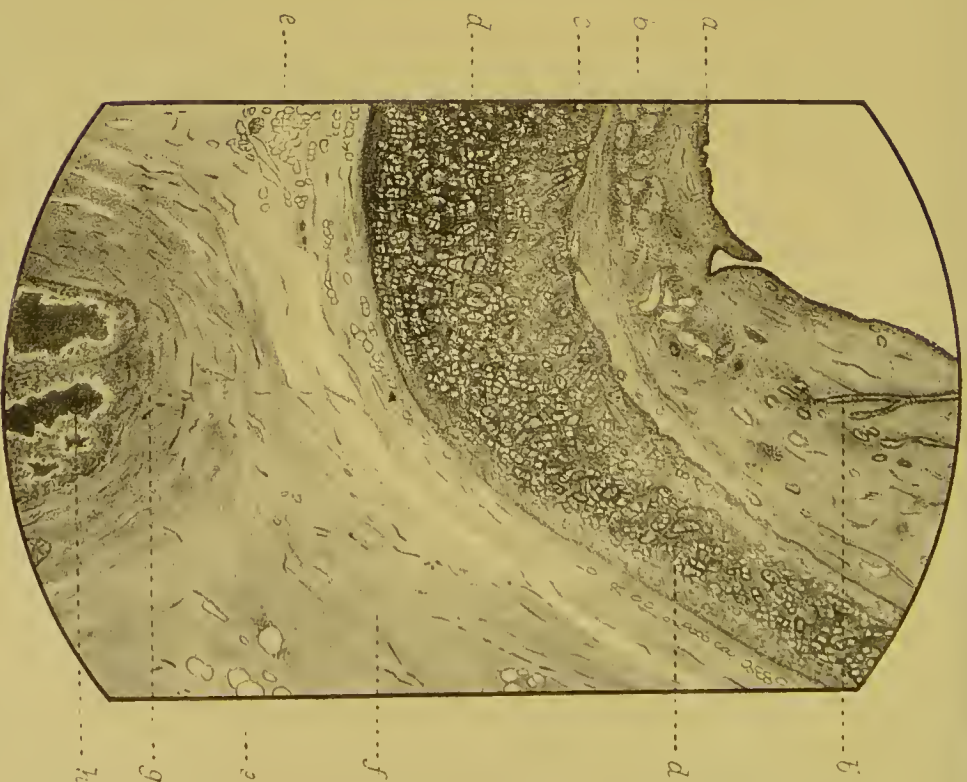


Fig. 2.

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